Society Reports.

NEW YORK NEUROLOGICAL SOCIETY.

Meeting of March 3, 1891.

The Vice-President, DR. B. SACHS, in the Chair.

AN HEREDITARY TYPE OF MOTOR TABES.

DR. PHILIP COOMBES KNAPP, of Boston, read a paper with this title. The case forming the subject of his remarks was one of muscular atrophy, occurring in a man thirty-four years of age. The trouble had commenced with fibrillary twitchings in the muscles of the right thigh, followed by weakness and atrophy. There were no sensory disturbances. Faradic excitability was lost and galvanic excitability diminished, and the contractions were slow, but there was no change on formula. The disease had progressed steadily, both legs becoming helpless and atrophied. The fibrillary twitchings finally appeared in the muscles of the shoulder and upper arm. There was a history of a kick in the right thigh, not of a very serious character, a few months before the onset of the symptoms. There was also a history of a peculiar affection in the patient's mother's family, of which the mother and at least four others in preceding generations had died. The affection was marked by paralysis of all the limbs, and in no case had there been atrophy. All the attacks had come on in middle life. The case reported was regarded as of spinal origin, and it was thought that the family disease was also spinal. In connection with this case, an instance of typical Aran-Duchenne atrophy, beginning in the thumb muscles, was reported, the patient's brother having died of a similar trouble. A case of progressive atrophy with bulbar symptoms was also cited, dating from a fall on the elbow.

DR. W. R. BIRDSALL said that the history detailed in the paper of Dr. Knapp had brought to his mind numerous cases of traumatic tabes, in which accident had developed some local, sensory or motor neuroses, a good many years before the usual types of symptoms of tabes were determined. Still, he did not think that the pathology of these cases was at all clear. There was little doubt, however, that a slight traumatism might form the point of origin where there already existed a family taint or a condition of the spinal cord favorable to the development of tabes. In the case cited by the author of the paper there were indications approaching traumatic tabes and time might develop other symptoms of myelitic disease.

DR. E. D. FISHER admitted that the injury might have had its influence. The hereditary cases were likely to have a mixed form of spinal disease with motor symptoms of involvement of the posterior columns with the class of symptoms of sensory tabes. Such a case as the one reported might be looked upon as one of hereditary disease.

The CHAIRMAN said that while the suspicion of hereditary trouble in the author's case was great, still the traumatism was a factor which could not be overlooked; and this, with the hereditary predisposition, might have acted as an exciting cause. The symptoms pointed to the myelopathic form of the disease, with fibrillar tremor and characteristic degeneration. They had all seen cases of progressive muscular atrophy which had begun in the leg without any hereditary history. With further observation of the case under discussion, the question could be tested whether the symptoms presented any new type of the hereditary form.

DR. KNAPP thought the question of sensory tabes in his case was hardly worth consideration, the occasional pains in the leg being the only sensory symptoms.

A CASE OF SPASTIC PARAPLEGIA. COMPENSATORY GAIT.

DR. BIRDSALL presented a child with a history of spastic paraplegia, which had supervened upon a severe instrumental delivery at birth. He had thought the case of interest because, while the child was only affected on the right side, which caused it to walk upon the toes of the affected foot, it so adjusted the position of the other foot as to secure a kind of compensatory gait, which enabled it to get about with more or less facility.

DR. F. PETERSON said the case appeared to him to exhibit symptoms of hemiplegia of one side and crural hemiplegia of the other side, which Dr. Birdsall considered was unaffected, or in other words, a partial diplegia. There was certainly considerable adductor spasm, with cross-legged position and exaggeration of reflexes, with contractures of the feet. He had seen a large number of cases of spastic paraplegia, in which one arm was involved, in addition, but without entire diplegia.

The CHAIRMAN thought that the child had sufficiently marked symptoms of diplegia, except that the left arm and the left leg were not much involved. The knee-jerks were exaggerated on both sides. It seemed to him that the difference between the two sides was that the contractures on the left side had relaxed and that the child had consequently learned to use the leg a little better than the other.

CLINICAL AND PATHOLOGICAL OBSERVATIONS ON INJURIES OF THE CERVICAL AND SPINAL CORD.

This was the title of a paper by DR. C. A. HERTER. The histories of five cases of injury to the spinal cord was detailed. The first four were all examples of severe crushing; the fifth was an instance of injury to the cervical vertebræ, with relatively slight damage to the nervous structures in this region. Among the most interesting features of these particular cases were their bearings upon the localization of the functions of the cervical cord. In Case I. there was an upward extension of the motor paralysis, from the interossei and flexors of the fingers, to the extensors of the fingers and wrists, the pronators and supernators, the triceps, biceps, and deltoids, successively. In Case II. the order of advance was much the same. In both cases the anæsthesia occupied the body and legs below a V-shaped line across the upper part of the chest, and the inner half of the arms, forearms and hands. While the motor symptoms progressed upward in both cases, the area of anæsthesia made no advance. When the patients were first seen, they presented essentially the same motor and sensory phenomena, namely: weakness of the hand muscles, and the distinctive anæsthesia above mentioned. The cord-lesion was the same in both cases, *i.e.*, complete crushing at the eighth segment, and partial softening of the seventh and sixth segments. There was no doubt that the peculiar distribution of the anæsthesia was due to the crush of the eighth segment. In both cases the bone lesion was a fracture dislocation of the sixth upon the seventh cervical vertebra. In three of the four cases, there was pressure of the displaced or fractured vertebræ upon the cord at the time of the operation or autopsy. In all total transverse lesions of the cord, and especially in those of the cervical and lumbar enlargements, certain symptoms were referable to the damage of the cord as a central organ, as opposed to those symptoms which depended on the obliteration of the functions of the cord as a conductor of impulses. These symptoms in the cervical region included loss of power and cutaneous sensibility, muscular atrophy, and degenerative electrical reactions. In two of the cases (IV. and V.) there were abdominal symptoms worthy of note. In each case, on the day after admission, the abdomen became tympanitic, exceedingly tender to pressure, and repeated vomiting occurred, the vomitus being at one time of a greenish color. The abdominal distention became very great, but began to subside, together with the pain and tenderness and vomiting, in the course of a few days. The temperature in cases of injury to the cervical cord varied much according to the severity of the damage. The last feature of these cases to which it was desired to call attention was the state of the reflexes, especially that of the knee-jerks. The superficial reflexes, cremasteric, plantar and abdominal were commonly lost from the beginning, in cases of complete crush of the cord, but a day or two sometimes passed before they were all lost. If the patients survived for several days or a week, some or all of the superficial reflexes returned. In all four of the cases of complete crush of the cervical cord, the knee-jerks were abolished, from a very early period after the accidents which caused these crushes respectively. The motor and sensory paralyses below the level of the lesion were complete, and in all the termination was fatal. It might safely be concluded that when bilateral loss of the knee-jerks follows immediately upon a sudden lesion of the cervical cord, we had to deal with a case of extensive and total transverse damage to the cord, and that the patient would die from it. It was not urged, of course, that this diagnostic and prognostic sign should be used to the exclusion of other associated conditions. These considerations led to one important practical conclusion, namely, that cases of crush of the cord, presenting complete or considerable paralysis of motion and sensation below the lesion and associated with loss of knee-jerks, should not be operated upon.

DR. KNAPP said that the point brought forward by the reader of the paper upon the question of the inhibitory suppression of the knee-jerk was of interest, as he had advanced the same idea many years ago. His case had been one of transverse myelitis, but not of traumatic origin. Entire absence of knee-jerk had been observed. Some weeks after, when the irritative processes had ceased in the lower portion of the cord, there were very pronounced exaggerations of the knee-jerks, with ankle and patella clonus.

DR. FISHER said it was a question whether, if the lesion was not destructive, but simply of an irritative character, there would be a loss or an exaggeration of the reflexes. He thought that the irritation must be very severe indeed to give rise to loss of knee-jerk.

The CHAIRMAN said that the phenomena connected with the loss of knee-jerk were quite different in cases of chronic cervical injuries. Its sudden abolition was entirely restricted to these cases of traumatism.

DR. HERTER said that cases were on record of slow inflammatory processes resulting in loss of knee-jerk, but he had never seen such a case.